CHRONIC SOCIAL STRESS AND CARDIOVASCULAR RESPONSE TO CONCENTRATED PARTICULATE AIR POLLUTION (CAPS) IN RATS

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Background and Aims: Epidemiologic evidence suggests that chronic stress may alter susceptibility to air pollution. Persistent spatial confounding between these exposures, however, limits the utility of epidemiologic methods alone to disentangle these effects, and it can not identify physiologic mechanisms.
Methods: Using a rat model of social stress, we examine continuous respiratory and cardiovascular responses to concentrated fine ambient particles (CAPs). Twenty-four 12-week old male Sprague-Dawley rats are randomly assigned to four groups (Stress/CAPs; Stress/Filtered Air (FA); Non-stress/CAPs; Non-stress/FA). Stress group animals are individually introduced into a dominant male's home cage twice weekly. Blood drawn at sacrifice is analyzed for immune and inflammatory markers.
CAPs are generated using the Harvard fine particle concentrator, which enriches real-time urban ambient fine particles by approximately a factor of 30. CAPs/FA exposures are delivered in single-animal plethysmographs, 5 hours/day on 10 days, with respiratory function continuously monitored using a Buxco system during CAPs exposures. Cardiovascular measures are collected via telemetry during CAPs and stress exposures. Lungs, hearts, and adrenals are fixed at constant pressure and random slices obtained for histology.
Results: We previously reported that stressed animals display higher average CRP, TNF-alpha, and white blood cell counts. In response to CAPs, stressed animals display increased respiratory frequency, lower flows and volumes -- suggesting rapid, shallow breathing patterns. Preliminary cardiovascular data indicate elevated heart rate (HR), systolic and diastolic blood pressures (SBP, DBP) during stress exposures.
Conclusions: Our findings suggest that chronic stress may alter respiratory and cardiovascular responses to air pollution in animals. Higher CAPs exposures were associated with a rapid shallow breathing pattern, only under chronic stress. Stress exposures also induce increases in HR, SBP, DBP, and temperature. Blood measures point to possible inflammatory pathways. Preliminary analyses suggest stress-related alterations in CAPs response; further investigation is needed to identify possible physiologic pathways for differential susceptibilities.